444 SERUM PROLACTIN IN INTRAVENTRICULAR HEMORRHAGE (IVH). T. Layaoen, R. Jhaveri, W. Rosenfeld, F. Khan, H. Evans, Department of Pediatrics and Medicine, Interfaith Medical Center and Downstate Medical Center.

Prolactin has been thought to play a role in preventing mobilization of tissue water in the neonate. Serum levels of prolactin are low in premature newborns and this may lead to decreased intracellular water, brain shrinkage, capillary bleeding and subsequent IVH. The hypothesis that decreased prolactin levels contribute to development of IVH was studied by our group.

Sixteen premature neonates $\leq$ 32 weeks gestational age with a mean birthweight of 1125 gms were followed prospectively with serial neurosonograms for the development of IVH. Serum prolactin levels by RIA were analyzed from cord blood and on days 1, 3, 7, 10, and 14.

Five neonates ($\bar{m}$ GA = 29.2 wk; $\bar{m}$ wt 1095 gms) with no IVH or gr I-II IVH were compared to 11 neonates ($\bar{m}$ GA 29.4 wk; $\bar{m}$ wt 1139 gms) who developed gr III-IV hemorrhage.

Mean serum prolactin levels (ng/ml) were:

IVH	CORD	DAY 1	3	7	10	14
gr 0, I, II	82	92	58	50	64	71
gr III, IV	63	81	50	51	55	56

Serum prolactin levels were comparable between the 2 groups. Our results do not support the concept of a pathogenetic role for prolactin in development of IVH.

445 LOW DOSE ORAL CLONIDINE: A SIMPLE AND RELIABLE GROWTH HORMONE (GH) SCREENING TEST. Roberto Lanes, Bridget Recker, Pavel Fort and Fima Lifshitz. Department of Pediatrics, North Shore University Hospital, Manhasset, NY 11030, and Department of Pediatrics, Cornell University Medical College, New York, NY 10021.

Clonidine has been shown to be an effective GH releasing agent in prepubertal children at a dose of 150 micrograms/M^2 body surface area, but at this dose it can produce significant side effects. We have evaluated the efficacy and side effects of low dose oral clonidine on GH, cortisol and blood pressure in 24 healthy short children; ten received 100 micrograms (Group A), and fourteen 50 micrograms (Group B). A 100 micrograms of oral clonidine induced higher, more sustained GH levels and fewer failures to testing than did 50 micrograms. The mean $\pm$ SD GH peak at 60 minutes in group A was 14.5 $\pm$ 6.3 vs 11.6 $\pm$ 6.1 ng/ml in group B (NS). Failure rate (GH < 10 ng/ml) was 10% in group A and 36% in group B. Children who responded to clonidine in group A (GH$\geq$10 ng/ml) had significantly higher GH levels than did group B responders ($p < 0.01$). A similar, but significant drop in cortisol was seen in both groups (from 14.5 $\pm$ 6.3 to 7.8 $\pm$ 2.6 ug/dl in group A and from 16.9 $\pm$ 6.7 to 12.8 $\pm$ 8.4 ug/dl in group B). Blood pressure did not change significantly with either dose used and only mild somnolence was noted in both groups.

A single oral dose of 100 micrograms of clonidine, followed by 1 blood sample 60 minutes later can be used to effectively and safely screen short children for GH deficiency.

446 POSSIBLE 11-HYDROXYLASE DEFICIENCY ADRENAL HYPERPLASIA AMONG VIRILIZED YOUNG FEMALES. P.A. Lee[1], R. Lanes[2], M.D. Urban[3], Departments of Pediatrics, [1]Children's Hospital of Pittsburgh, Pittsburgh, PA; [2]North Shore University Hospital, Manhasset, NY; [3]Children's Medical Center, Dayton, OH

To determine if plasma desoxycortisol (Compound S-Cmpd S) basal and ACTH-stimulated response levels allow identification of patients with mild 11-hydroxylase deficiency, this hormone has been measured in the following 4 groups of subjects: (1) females presenting with premature pubarche or hirsutism in adolescence; (2) subjects who are heterozygotic carriers or have mild 11-hydroxylase deficiency based on kindred studies; (3) normal females, post menarchial and follicular phase; (4) 10 females known to be heterozygotic carriers of 21-hydroxylase deficiency.

		Basal Cmpd S Levels (ug/dl)			Incremental Rise (ug/dl) 30 min post 100u ACTH IV		
	#	Mean	S.D.	Range	Mean	S.D.	Range
1) Unknown	22	78.1	65.0	12-226	118.1	69.6	2-256
2) 11 OH Def	9	37.8	33.6	10-106	199.1	135.8	103-527
3) Normal ♀	9	56.7	43.8	15-136	69.6	53.2	4-131
4) 21 OH Def	10	17.8	8.6	10- 34	176.4	89.2	85-316

Seven of the females with excessive hair for age (1) had Cmpd S responses greater than normal controls (3), but within the range of those with evidence of compromised 11 OHase (2) or 21 OHase (4) action. Five of these seven had basal levels greater than Group 4. These patients could have mild 11-hydroxylase deficiency.

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● 447 SERUM 1,25-DIHYDROXYVITAMIN D IN INFANCY: SEASONAL & RACIAL DIFFERENCES, Philip Lichtenstein, Bonny Specker, Charles Gormley, Kay Ellis, Reginald C. Tsang, U. of Cincinnati College of Medicine.

Serum 1,25-dihydroxyvitamin D (1,25(OH)$_2$D$_3$), the active hormone principle of vitamin D, is finely regulated in adults. 1,25(OH)$_2$D levels are elevated in infancy relative to adulthood possibly reflecting increased Ca and P needs during periods of active bone growth. There is little information on factors affecting 1,25(OH)$_2$D concentrations in infancy. Effects of diet & race have not been examined. We examined the hypothesis that serum 1,25(OH)$_2$D would not be affected by 1) breastfeeding vs formula feeding, 2) season, and, 3) race. 143 normal infants less than 18 months of age were examined. 1,25(OH)$_2$D was measured by Eisman's method (HPLC-cytosol binding protein, normal 17 to 44 pg/ml). Infant values were elevated vs adults ($p < .001$). By analysis of variance, no significant difference was found between breast-fed (58 $\pm$ 4 pg/ml, mean $\pm$ SE) and bottle-fed infants (66 $\pm$ 4 pg/ml). Significant seasonal and racial differences were found, with winter infants having higher 1,25(OH)$_2$D$_3$ (72 $\pm$ 3) than summer infants (53 $\pm$ 3, $P < .001$), and black infants (70 $\pm$ 3) having higher concentrations than white infants (56 $\pm$ 2, $p < .001$). Seasonal differences remained within subgroups: in whites wnter mean was 62 vs summer 50 pg/ml; in blacks winter 81 vs summer 56 pg/ml. No age-specific effects were apparent within the age range studied. Thus, serum 1,25(OH)$_2$D concentrations in infants < 18 months of age are not affected by breast vs formula feeding, and are increased in winter & in blacks. We speculate that differences in 1,25(OH)$_2$D concentrations may be sensitive indicators of vitamin D status in infancy and should be taken into consideration when studies of vitamin D and calcium metabolism are undertaken in infants.

448 ACUTE AND CHRONIC EFFECTS OF hGH ON LIPOPROTEINS IN CHILDREN WITH GROWTH HORMONE DEFICIENCY. Tsu-Hui Lin, Rebecca T. Kirkland, Jeffrey L. Probstfield, William Insull, Bruce S. Keenan, George W. Clayton. Baylor College of Medicine, Depts. of Ped. and Med., Houston, TX.

Ten prepubertal Growth Hormone deficient subjects, aged 2 - 13 6/12 years old, 3 females and 7 males (3 F, 7 M), received 0.1 u/kg human GH (hGH) daily with lipoproteins (HDL-C) measured before and at the end of 4 days of hGH. Nine patients received long term hGH 0.1 u/kg 3 times per week with HDL-C measured at 3 months, 6 months and 12 months. The diagnoses were: craniopharyngioma, 4; idiopathic GH deficiency, 4; and septo-optic dysplasia, 2. Ten patients completed the acute study; 9 patients, (3 F and 6 M) completed 3 months; 7 patients, (2 F and 5 M) completed 6 months, and 5 m completed the 12 month studies. All subjects but one had a decrease in the HDL-C (mean $\pm$ S.E. 50.1 $\pm$ 4.0 to 41.8 $\pm$ 2.9, $p < 0.025$ - paired t test) in the acute phase. In the chronic study no significant change of HDL-C was noted before and at 3 months, 6 months or 12 months after hGH treatment. (mean $\pm$S.E. 50.1 $\pm$ 4.0, 49.8 $\pm$ 3.0, 50.0 $\pm$ 3.4 and 47.2 $\pm$ 5.9 mg/dl) respectively. Testosterone levels did not change (10 subjects).

The mechanism of this acute decrease in HDL-C is unclear but could be explained by altered bile acid metabolism, apoprotein A-1 metabolism, effects mediated by other hormones induced by GH, or combination of these. The acute decrease in HDL-C and the absence of a significant change with chronic hGH observed in this study may explain some of the conflicts reported by other investigators.

† 449 EFFECT OF LOW DOSE PREMARIN AND HALOTESTIN ON LIPOPROTEIN IN TURNER SYNDROME. Tsu-Hui Lin, Rebecca T. Kirkland, Jeffrey L. Probstfield, Sheryl A. Hausinger, William Insull, Bruce S. Keenan, George W. Clayton. Baylor College of Medicine, Depts. of Ped. and Med., Houston, TX.

Sex hormones have been shown to alter high density lipoprotein cholesterol levels (HDL-C). Estrogens increase, while androgens (A) decrease, HDL-C. Some progestogens reduce HDL-C, but effects of low dose combined oral contraceptives remain controversial. Data is sparse concerning the effect of low dose A therapy. 11 patients with Turner syndrome (TS), aged 9 to 14 years, received Premarin (P, conjugated estrogens) 0.15 mg (0.16 $\pm$ 0.004 mg/M^2) and Halotestin (H, Fluoxymesterone) 1 mg (1.09$\pm$0.03 mg/M^2) p.o. daily with the exception of one girl who received P,H every other day. Baseline HDL-C was measured after at least 3 months without P, H and after 6 and 12 months of therapy. All 11 girls completed 6 months and 5 girls 12 months of therapy. Mean $\pm$ S.E. levels of HDL-C for baseline, 6 and 12 months were 50.7 $\pm$ 1.9, 45.0$\pm$ 2.8 and 45.0 $\pm$ 2.8 mg/dl respectively and indicated significant decrease from baseline to 6 month ($p < 0.01$ - paired t) and 12 month ($p < 0.025$ paired t) of therapy. Plasma testosterone, measured in 9 girls was in the prepubertal range. In 3 TS girls, 15, 12, 9 years, receiving low dose P without H for more than 6 months, HDL-C levels were 59, 49 and 56. Our study suggests that even low dose H therapy can reduce HDL-C, and may imply that low dose combined oral contraceptives can be associated with reduced HDL-C in normal females. Girls with TS or other disorders who receive A may have decreased HDLC which has been associated as an independent risk factor in coronary heart disease.